Case Report

Headache Angina with Fatal Outcome

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SUMMARY

We report 2 fatal cases of angina pectoris in patients who complained primarily of headache during the ischemic attack. The first patient, who was hospitalized because of headache and chest pain, demonstrated repeated ST-segment elevation and fatal ventricular fibrillation on ambulatory ECG monitoring. The second patient had post-infarction angina preceded by headache and by ST-segment elevation in the precordial leads. She eventually died of reinfarction. The mechanism of the headache in relation to the angina pectoris is discussed.

Additional Indexing Words:

Neurological symptoms Vasospastic angina Myocardial infarction
Ventricular fibrillation

HHEADACHES have been known to often precede or accompany attacks of angina pectoris.¹⁾ A recent report comparing variant angina to classic angina and control groups showed that the prevalence of headaches was significantly higher in the vasospastic angina group than in the other 2 groups.²⁾ However, few cases of headache angina have been described with precise ECG findings.³⁾ We report 2 fatal cases with headache angina. In each case, ECG monitoring during the attack and the effect of nitroglycerin were documented. In one of them, the fatal clinical course of headache angina was recorded on an ambulatory ECG and in the patient's diary.

CASE REPORTS

Case 1: A 67-year-old man came to our outpatient clinic complaining of headache and chest pressure on January 18, 1982. From January 12, he had a severe headache and vague chest pressure while taking a hot bath for

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Fig. 2. Case 2; Panel A: ST-segment elevation and peaked T waves were observed in leads V_1 to V_3 where the deep Q waves were seen during the angina following headache at 10 pm on February 11, 1988. ST-segment depression was seen in lead V_5 . Panel B: After 10 min, ST-segments became normalized in most of the leads following intravenous infusion of 0.3 mg isosorbide dinitrate.

intervals. The patient was asymptomatic until he awoke with a headache and chest pressure at 3:50 am. The ST-segment elevation was suppressed by sublingual nitroglycerin taken at 4:20 am and appeared again from 5:15 am. Thereafter, he described two attacks of headache angina until 6:25 am, when he stood up to urinate. ST-segment elevation disappeared from 7 am for 1 hour. The final event started with gradual ST-segment elevation as shown in Fig. 1-B from 8:07 to 8:08 am. ST-segment elevation reached a maximum at 8:09 am. During the recovery phase, a short run of ventricular premature beats suddenly turned into ventricular fibrillation at 8:10 am.

Case 2: A 64-year-old woman was hospitalized on December 8, 1987 because of increasing frequency of chest pain. One year before admission, she had an anterior myocardial infarction with severe three vessel disease,

for which coronary angioplasty was performed 4 times. Although the angioplasty dilated the anterior descending coronary artery from 95% stenosis to 50% and the right coronary artery from 99% to 75%, those vessels re-stenosed. From the beginning of February 1988, she felt frequent headaches which preceded the chest pain by several minutes.

ECG recordings during an episode of chest pressure following a headache on February 11 revealed ST-segment elevations in leads V_2 and V_3 , where deep Q waves were observed (Fig. 2-A). Ten minutes after intravenous administration of 0.3 mg isosorbide dinitrate, the pain and ST-segment elevation disappeared (Fig. 2-B). To relieve the anginal attack, parenteral nifedipine 80 mg/day or continuous nitrate infusion up to 0.6 mg/hour became necessary. She could suppress the headaches by prophylactic use of nitroglycerin. She had two subsequent attacks of acute myocardial infarction and eventually died of refractory heart failure in June, 1988. Autopsy revealed acute and healed anteroseptal myocardial infarctions with marked atherosclerosis of the left and right epicardial coronary arteries. The lumens of the coronary arteries on which coronary angioplasty had been performed showed moderate dilation.

DISCUSSION

Although headache and angina pectoris have both been highly suspected of being manifestations of generalized vasospasm, identification of the spasm was difficult in the reported cases.³⁾⁻⁶⁾ In our first case, frequent ST-segment elevations were the evidence of coronary arterial spasm. At midnight, coronary arterial spasm seemed to have preceded the headache, and both could be relieved by sublingual nitroglycerin. However, the effect of the nitroglycerin did not last long enough. One hour later, the patient had two attacks of headache angina with concurrent ST-segment elevation. The fatal attack, which turned into ventricular fibrillation, was possibly due to a reperfusion arrhythmia following coronary arterial spasm. In our second case, the patient died of heart failure with extended myocardial infarction. Spasm of the left anterior descending coronary artery was suspected from the ECG as a cause of her chest pressure. A coronary angiogram during the attack would have established a definite diagnosis. It was noted that her headache could be also prevented by prophylactic use of sublingual nitroglycerin.

From these findings, we can speculate on the clinical significance of neurological symptoms accompanying angina pectoris. Whether the headache was due to a referred pain transferred through the spinal cord following myocardial ischemia or due to a coincidental spasm of the cerebral artery

was not clear in spite of the effectiveness of nitroglycerin in both cases.⁵⁾⁻⁷⁾ Another limitation of our study is the lack of an objective description of the headaches. Quantification of headache in combination with the ECG monitoring would be required.

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